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Ultrasound of the inferior vena cava for fluid therapy decisions: Strengths, limitations, and an integrated approach

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Abstract

Accurate assessment of fluid responsiveness is critical in resuscitation; however, static indices often fail to predict it accurately. Inferior vena cava ultrasonography is valued for its simplicity and accessibility; however, its predictive accuracy in real-world settings remains uncertain. This narrative review examined the physiological rationale, limitations, and clinical utility of inferior vena cava ultrasonography. Although inferior vena cava ultrasonography leverages the Frank–Starling mechanism and heart–lung interactions, its measurements are susceptible to confounders (e.g. ventilator settings, spontaneous breathing, right-heart dysfunction, and intra-abdominal pressure) and operator-dependent variability. Evidence from heterogeneous cohorts has shown moderate accuracy, with reliable performance often limited to strictly controlled conditions. In lung-protective ventilation or mixed populations, its discriminative ability declines, raising the risk of misclassification. Consequently, inferior vena cava ultrasonography should not be used as a standalone tool for guiding fluid therapy. Its optimal role lies within a multimodal, iterative strategy incorporating passive leg raising, left ventricular outflow tract velocity–time integral, lung ultrasound, and venous excess ultrasound. In practice, inferior vena cava ultrasonography

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should inform but not decide fluid therapy; its safest role is as a contextual trend within a physiology-first, function-centered, and congestion-aware workflow.

Keywords
Inferior vena cava, ultrasonography, fluid responsiveness, hemodynamic monitoring, functional hemodynamic tests, end-expiratory occlusion, tidal-volume challenge, left ventricular outflow tract velocity–time integral, lung ultrasound, venous excess ultrasound

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Introduction

Accurate assessment of fluid responsiveness is a cornerstone of critical care, balancing adequate tissue perfusion with the risk of fluid overload, which is associated with increased morbidity and mortality.[1,2] Traditional static indices, such as central venous pressure (CVP) and clinical examination, poorly predict fluid responsiveness.[3] Consequently, guidelines advocate dynamic functional tests, including passive leg raising (PLR) with cardiac output (CO) or left ventricular outflow tract velocity–time integral (LVOT VTI) monitoring, end-expiratory occlusion, and tidal-volume challenge.[3] No single ultrasonographic metric is universally endorsed for guiding fluid therapy decisions.

Inferior vena cava (IVC)–ultrasonography (US) has gained popularity due to its noninvasive nature, low cost, and repeatability.[4] As a compliant vein contiguous with the right atrium, the IVC's diameter and respiratory variation theoretically reflect preload changes, offering a bedside "window" into volume status.[5] Its short learning curve and accessibility have driven its adoption in emergency departments (EDs), intensive care units (ICUs), and similar settings.[6]

However, as methodological rigor has increased across studies, appraisals of its clinical utility have become more circumspect. Investigators such as Millington have critically challenged key assumptions underlying IVC–US and questioned its capacity to improve clinical outcomes, despite its intuitive appeal.[7] Accumulating evidence has indicated that IVC indices lack consistency and perform less reliably than anticipated in predicting fluid responsiveness, thereby contributing to the controversy. In this narrative review, we synthesized physiological rationale, technical constraints, and clinical evidence to offer clear, practice-oriented recommendations.

Literature search and scope (Scale for the Assessment of Narrative Review Articles (SANRA)-guided)

This narrative review followed the SANRA guidelines.[8] We searched PubMed, Embase, Web of Science, and Scopus (January 2010 to October 2025) using combinations of "inferior vena cava," "ultrasound," "fluid responsiveness," "collapsibility," "distensibility," "passive leg raising," "end-expiratory occlusion," "tidal-volume challenge," and "VExUS." Adult ICU/ED studies reporting accuracy (sensitivity, specificity, and area under the curve (AUC)), thresholds, or gray-zone analyses for IVC–US were included; pediatric studies, intraoperative TEE-only data, or highly selected disease-specific series were excluded. Two reviewers screened titles/abstracts and full texts; disagreements were adjudicated by a third author. Reference lists of key reviews were snowballed to identify additional studies.

Theoretical underpinnings and physiological rationale

Frank–Starling mechanism and preload assessment

IVC–US is grounded in the Frank–Starling mechanism, where CO increases with ventricular preload until a plateau is reached, beyond which additional fluids may be ineffective or harmful.[9] IVC–US aims to identify patients on the ascending limb of this curve where fluids enhance the CO. The IVC's compliance and proximity to the right atrium allow its diameter and respiratory variation to theoretically reflect preload changes. In hypovolemia, reduced venous return narrows the IVC and increases inspiratory collapsibility, while adequate or excess volume enlarges the IVC and reduces collapsibility.[6] However, these patterns are prone to confounding, complicating interpretation.

Respiratory physiology and IVC dynamics

Respiratory variations in the IVC diameter underpin its use in assessing fluid responsiveness. During spontaneous breathing, inspiration lowers the intrathoracic pressure, enhancing venous return and collapsing the IVC. During mechanical ventilation, positive-pressure inspiration increases intrathoracic pressure, reducing the venous return and distending the IVC.[10] Key metrics include the collapsibility index (CI) = [(IVC_max − IVC_min)/IVC_max] × 100%) for spontaneous breathing and the distensibility index (DI) = [(IVC_max − IVC_min)/IVC_min] × 100%) for mechanical ventilation.[7,11,12] These indices enable rapid bedside assessment but require careful interpretation based on the ventilation mode.

Limitations of the theoretical mode

Despite its physiological basis, IVC–US has significant limitations. The IVC diameter does not linearly correlate with intravascular volume and is influenced by venous compliance, which varies across individuals and conditions (e.g. heart failure and liver disease).[12] Preload is only one determinant of CO; contractility, afterload, and heart rate also play critical roles, and IVC–US cannot identify underlying causes of hypotension (e.g. distributive and cardiogenic shock). Nonspecific factors, such as respiratory effort, airway resistance, and intra-abdominal pressure (IAP), further decouple IVC variation from the volume status, particularly in conditions such as chronic obstructive pulmonary disease (COPD) and intra-abdominal hypertension.[13] Ventilation mode, arrhythmias, right-heart function, and sampling variability further disrupt the inferential chain from IVC metrics to preload reserve.[14–16]

Technical aspects and measurement challenges

Ultrasound technique and image acquisition

Accurate IVC–US requires standardized imaging, typically using a subxiphoid long-axis view with the probe angled toward the right shoulder. The 2015 American Society of Echocardiography (ASE)/ European Association of Cardiovascular Imaging (EACVI) guidelines recommend sampling 1–2 cm distal to the IVC–right atrial junction or hepatic vein confluence to minimize right atrial pressure wave interference.[17] Image quality depends on patient factors (e.g. obesity and bowel gas) and operator skill, with poor acoustic windows often compromising accuracy.[18]

Measurement variability and standardization issues

Inconsistent imaging standards contribute to IVC–US variability. Studies differ in

imaging mode (M-mode vs. 2D), anatomic target, and respiratory phase, reducing comparability.[19] Even expert operators exhibit inter- and intra-observer variabilities (coefficients of variation ≈15%–25%), driven by subjective plane selection, timing, and millimetric positional shifts.[15,19,20] Errors such as foreshortening and misidentification (e.g. mistaking the aorta for the IVC) further reduce reliability.

Technical artifacts and confounders

Nonvolume factors significantly affect IVC metrics. Arrhythmias introduce beat-to-beat variability, while tricuspid regurgitation (TR) decouples diameter changes from volume status.[17,21] Ventilator settings (e.g. positive end-expiratory pressure (PEEP) and tidal volume) alter intrathoracic pressure and IVC dynamics without reflecting true blood volume.[22] Spontaneous breathing, right-ventricular dysfunction, pulmonary hypertension, and elevated IAP further distort measurements.[16,23,24] Lung-protective ventilation (low tidal volumes and spontaneous effort) disrupts heart–lung interactions, limiting the utility of dynamic indices.[25,26] Factors influencing IVC measurements are summarized in Table 1.

Evidence summary: ventilation dependence and inconsistent thresholds

In ICUs, approximately 50% of patients with circulatory failure do not respond to fluids and may experience adverse outcomes.[27] Early studies have suggested that IVC–US is a viable noninvasive tool; however, these studies involved selected cohorts with strict conditions (e.g. controlled ventilation and high tidal volumes).[28,29] In spontaneously breathing patients, collapsibility reflects inspiratory effort more than preload, with a wide grey zone.[22,30] In mechanically ventilated patients, accuracy requires controlled ventilation, tidal volume $\geq$8 mL/kg, low PEEP, sinus rhythm, and preserved right-heart function—conditions often incompatible with lung-protective strategies.[22,24–26,31]

Meta-analyses highlight significant heterogeneity. Kim et al. (2021) reported

Table 1. Factors influencing IVC caliber.

Physiological factors	Clinical scenario	IVC change
Ventilator settings	High PEEP	Enlargement
Spontaneous inspiratory effort	Assisted ventilation	Enlargement or collapse
Lung hyperinflation	Wheezing, COPD exacerbation	Enlargement
Increased intra-abdominal pressure	Peritonitis, peritoneal inflammation, bowel obstruction, etc.	Enlargement or collapse
Right-atrial pressure and right-heart compliance	Right-heart dysfunction, tricuspid regurgitation	Enlargement
	Cardiac tamponade	
	Pulmonary hypertension	
Local factors	Impaired venous return (stenosis and thrombosis)	Enlargement
	Extrinsic compression of the IVC (mass)	Collapse
	Restricted IVC motion (ECMO cannula, IVC filter)	Enlargement or wall apposition (flattening)

PEEP: positive end-expiratory pressure; COPD: chronic obstructive pulmonary disease; ECMO: extracorporeal membrane oxygenation; IVC: inferior vena cava.

pooled sensitivity of 0.75, specificity of 0.83, and AUC of 0.86; however, the high heterogeneity ($I^2 = 84\%–91\%$) limits generalizability.[32] In 2018, Orso et al. reported a lower AUC of 0.71, concluding that IVC–US is unreliable as a sole guide.[30] Recent studies, such as those conducted by Dunfield et al. in 2023 (AUC 0.92) and El-Gazzar et al. in 2022 (AUC 0.43), further underscore inconsistent performance across settings.[33,34] IVC–US is best used as an adjunct within a multimodal framework.

Critical analysis of limitations

IVC–US reliability is undermined by its dependence on optimal positioning, acoustic windows, and stable respiratory mechanics—often unachievable in critically ill patients.[17] Positive-pressure ventilation complicates interpretation, and spontaneous breathing or respiratory disease can decouple IVC dynamics from volume status.[22,25,26] The technique cannot identify hypotension causes, risking inappropriate fluid administration.[23,24,35–38] Operator variability (15%–25%) and the need for structured training further limit reproducibility.[15,19,20] Thus, IVC–US should complement, not replace, comprehensive hemodynamic assessment.

Alternative approaches and comparative effectiveness

Dynamic parameters

Dynamic tests, such as stroke volume variation (SVV) and pulse pressure variation (PPV), outperform static measures such as IVC diameter, with AUCs often exceeding 0.85 in ventilated patients.[37,38] PLR, a non-invasive functional test, accurately predicts responsiveness across ventilation modes by monitoring CO or LVOT VTI changes.[38] Mini-fluid challenges (100 mL crystalloid with ΔVTI $\approx$10%) offer a pragmatic alternative when PLR is infeasible.[39]

Integrated hemodynamic assessment

A multiparametric approach addresses both responsiveness ("can CO increase?") and tolerance ("can the patient tolerate fluids?").[25]

1. *Echocardiography with VTI/CO trending*. Rapidly profile systolic/diastolic and right-heart function and volume status; use dynamic VTI/CO change as the responsiveness readout.[25,40]
2. *Lung ultrasound (LUS)*. B-lines and pleural effusions indicate pulmonary fluid burden and intolerance; rising B-line counts during fluids are a "brake signal."[41]
3. *Venous excess ultrasound (VExUS)*. Integrates IVC with hepatic, portal, and intrarenal venous Doppler to grade systemic venous congestion and acute kidney injury (AKI) risk, helping in identifying when to stop fluids.[23,42]
4. *Context gating*. Ventilation-dependent indices (ΔIVC/PPV) perform acceptably only when multiple prerequisites are met simultaneously—controlled ventilation, tidal volume (VT) $\geq$8 mL/kg, low PEEP, no spontaneous effort, sinus rhythm, preserved RV function; under lung-protective ventilation (VT $\leq$6–8 mL/kg), prioritize PLR or a tidal-volume challenge.[25,37,38]
5. IVC metrics are one component of an evidence bundle; combined with echocardiography, LUS, and VExUS, they improve the safety of fluid therapy.

Clinical implications and recommendations

Risk–benefit analysis

The use of IVC–US should be appraised within a strict risk–benefit framework: the test is safe and accessible; however, the downstream treatment decisions it triggers

can profoundly affect outcomes. False positives—readings that suggest the presence of fluids despite nonresponsiveness—drive positive fluid balance, increase pulmonary edema, prolong mechanical ventilation, and raise mortality. False negatives—signaling nonresponsiveness in would-be responders—result in under-resuscitation and organ hypoperfusion. Together with wide grey zones and substantial population/technical heterogeneity, IVC metrics often lack discriminative power where precision is most needed; therefore, their real-world clinical benefit is well below early expectations.[22,35,37,38,43]

Appropriate clinical use

IVC measurements retain marginal value at the extremes (e.g. a very small, highly collapsible vein or a markedly dilated, noncollapsible vein), which can aid ED triage or care in resource-limited settings.[44] Where invasive monitoring is unavailable (ED triage or resource-limited settings), the IVC can serve as an adjunct for rapid stratification and cross-checking of prior clinical judgements. However, in heterogeneous contexts—critical illness and perioperative settings—the IVC should not be the sole determinant of fluid therapy; even as an adjunct, its prerequisites and limitations must be stated explicitly, preventing its use as a "direct-read gauge" of preload or responsiveness.

Integration with clinical decision-making

IVC readings must be integrated with history, examination, laboratory data, imaging, and point-of-care ultrasound (POCUS) and interpreted within the physiologic context as ventilation mode, tidal volume, PEEP, rhythm, right-heart function, and IAP can modify the interpretation.[14–17] When IVC findings conflict with the clinical picture, the results should be interpreted contextually and confirmed using more robust dynamic tests—PLR with real-time CO/VTI or a mini-fluid challenge targeting $\Delta VTI \approx 10\%$—and paired with LUS and VExUS to determine tolerance/congestion, rather than relying on a single IVC threshold.[25,40,41]

Future directions and research needs

Methodological improvements

Future investigations of IVC–US should first address previous methodological shortcomings (Figure 1). First, measurement workflows should be standardized—the anatomic sampling site, respiratory phase (or breathing-control strategy), measurement timing, and acquisition/reading protocols should be defined—to enhance between-study comparability and generalizability. Second, the IVC should be repositioned as secondary evidence integrated with LUS, focused echocardiography (including VTI/CO), functional tests (PLR/mini-fluid challenge), and venous congestion grading (VExUS) to mitigate iatrogenic harm from over-resuscitation within a responsiveness $\times$ tolerance framework.[3,36,37] Third, large, multicenter, methodologically rigorous, adequately powered studies enrolling diverse, real-world populations rather than preselected "ideal" participants should be conducted. Fourth, automated measurements and AI-assisted interpretation systems should be developed to curb interobserver variability, with thorough prospective validation and external calibration before clinical deployment. Fifth, a critical gap persists: no patient-outcome–level evidence has shown that IVC-guided fluid management outperforms standard care. Future work should prioritize patient-important outcomes—mortality, organ function, length of stay, quality of life—rather than relying solely on surrogates such as CO/VTI.

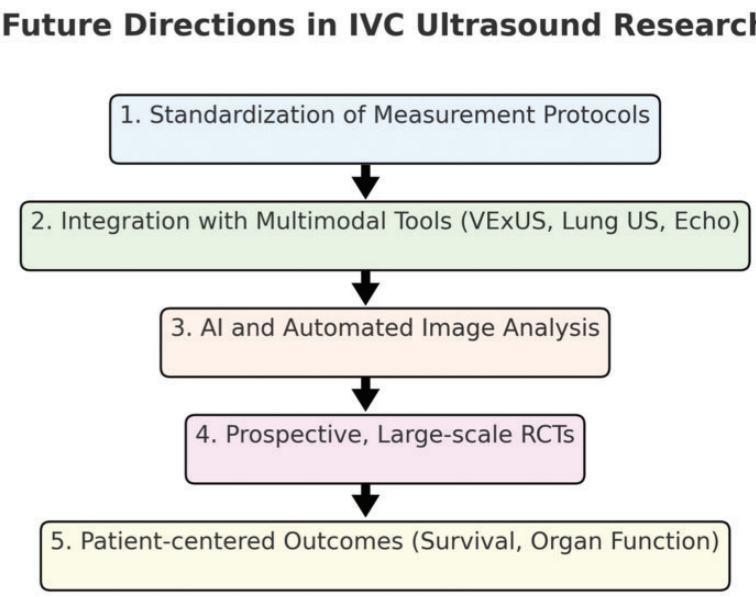
Future Directions in IVC Ultrasound Research
1. Standardization of Measurement Protocols
2. Integration with Multimodal Tools (VExUS, Lung US, Echo)
3. AI and Automated Image Analysis
4. Prospective, Large-scale RCTs
5. Patient-centered Outcomes (Survival, Organ Function)

Figure 1. Future directions in IVC ultrasound research. Conceptual roadmap highlighting context-aware thresholds (ventilation, RV function, and intra-abdominal pressure), validation of venous congestion staging across heterogeneous cohorts, and patient-centered outcome trials of integrated pathways. IVC: inferior vena cava; FR: fluid responsiveness; LUS: lung ultrasound; VExUS: venous excess ultrasound; PLR: passive leg raising; VTI: velocity–time integral; CO: cardiac output; RV: right ventricle.

Although current evidence is sufficient to support an integrated IVC+ approach in practice, prospective, context-standardized studies—ideally including patient-centered outcomes—would help refine thresholds, reduce variability, and assess health-economic impact. In parallel, the real-world incidence and clinical consequences of IVC-driven misclassification (false positives/negatives) should be quantified systematically.

Strengths and trend-based use of IVC–US

IVC–US is accessible, repeatable, and can be rapidly learned, which favors triage and resource-limited contexts. Although absolute thresholds are unreliable across heterogeneous settings, trend information—captured during PLR or a 100–150-mL mini-fluid challenge and interpreted in combination with LVOT VTI/CO—can support decision-making. When ΔIVC and ΔVTI (≈10%) concur and LUS/VExUS do not show rising congestion, a cautious 250–500-mL bolus followed by immediate re-evaluation is reasonable. Conversely, divergence between IVC trends and functional output or worsening LUS/VExUS should act as a signal to halt fluids and prompt vasopressors/inotropes or de-resuscitation measures.

Integrated algorithm

We propose a five-step pathway (Figure 2) that is physiology-first, function-centered, and safety-anchored.

1. *Confounder clearance*. Systematically screen for RV dysfunction, severe TR, elevated IAP, obstructive pathology, pregnancy/obesity, and poor windows, and promptly identify and treat must-not-miss causes (e.g. cardiac

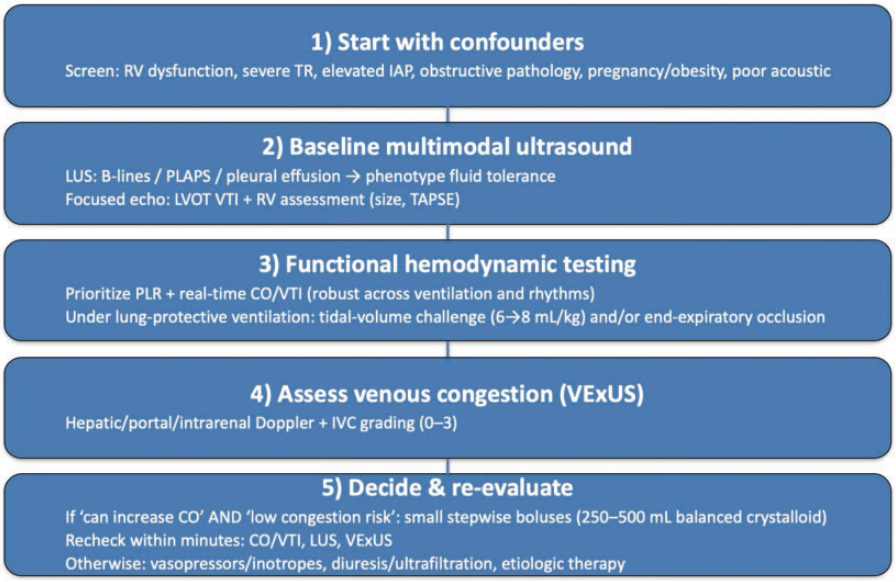
1) Start with confounders
Screen: RV dysfunction, severe TR, elevated IAP, obstructive pathology, pregnancy/obesity, poor acoustic
2) Baseline multimodal ultrasound
LUS: B-lines / PLAPS / pleural effusion → phenotype fluid tolerance
Focused echo: LVOT VTI + RV assessment (size, TAPSE)
3) Functional hemodynamic testing
Prioritize PLR + real-time CO/VTI (robust across ventilation and rhythms)
Under lung-protective ventilation: tidal-volume challenge (6→8 mL/kg) and/or end-expiratory occlusion
4) Assess venous congestion (VExUS)
Hepatic/portal/intrarenal Doppler + IVC grading (0–3)
5) Decide & re-evaluate
If 'can increase CO' AND 'low congestion risk': small stepwise boluses (250–500 mL balanced crystalloid)
Recheck within minutes: CO/VTI, LUS, VExUS
Otherwise: vasopressors/inotropes, diuresis/ultrafiltration, etiologic therapy

Figure 2. Integrated fluid management algorithm with IVC assessment. Five-step physiology-first, function-centered pathway: (a) confounders first; (b) baseline multimodal ultrasound (LUS; focused echo including LVOT VTI and RV assessment); (c) functional testing (PLR with VTI/CO; tidal-volume challenge or end-expiratory occlusion; mini-fluid challenge when justified); (d) venous congestion profiling (VExUS + IVC grading); and (e) small, reversible fluid trials with rapid reassessment. IVC: inferior vena cava; LUS: lung ultrasound; RV: right ventricle; VExUS: venous excess ultrasound; PLR: passive leg raising; LVOT VTI: left ventricular outflow tract velocity–time integral; CO: cardiac output.

tamponade and tension pneumothorax).[11,13,16]

2. *Baseline multimodal POCUS*. Use LUS to assess tolerance, focused echocardiography (VTI/CO) to assess pump function, and treat IVC findings as ancillary information.[15,16,23,26]
3. *Functional maneuvers*. Perform PLR with VTI/CO as the first-line test. If needed, perform a tidal-volume challenge or end-expiratory occlusion.[25,45–49]
4. *VExUS scoring*. Grade venous congestion to signal when to stop fluid administration.[23,50]
5. *Decision and iterative re-evaluation*. If responsiveness is high and tolerance is adequate, administer a small bolus (250–500 mL) and recheck; otherwise use vasopressors, diuresis, or targeted aetiologic therapy.[1,3,31,46]

Practice recommendations

IVC–US should not be used as a stand-alone determinant of fluid therapy. It is recommended to use PLR with VTI/CO as a first-line test across ventilation modes; in low tidal-volume ventilation, a tidal-volume challenge or end-expiratory occlusion should be additionally performed. Responsiveness testing should be paired with tolerance assessment via LUS and VExUS. IVC extremes and trend-based changes should only be considered as adjuncts within this integrated framework, with small, reversible fluid trials and rapid reassessment. Figure 2 depicts the five-step algorithm with IVC assessment. Within this framework, IVC indices should be treated as trend signals rather than fixed thresholds; action should be taken when ΔIVC

and ΔVTI/CO concur without any evidence of rising LUS/VExUS congestion. Small reversible boluses should be used with immediate re-evaluation, and fluids should be halted when trends diverge or congestion increases.

Conclusion

IVC–US is not a reliable standalone tool for fluid therapy due to its susceptibility to confounders and variability. Its value lies within a multimodal, physiology-driven strategy, integrating PLR/VTI for responsiveness, LUS/VExUS for tolerance, and iterative re-evaluation to optimize safety and efficacy in fluid management.

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All authors contributed to the critical revision of the manuscript.
Supervision: Xixi Guo, Yongwei Song, and Weiting Chen
Project administration: Yongwei Song and Weiting Chen
All authors read and approved the final manuscript and agree to be accountable for all aspects of the work.

Data availability statement

All relevant data are contained within the article.

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